Water and Electrolyte Disorders 2

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HYPERNATREMIA

= *WATER Deficit*

Hypernatremia: Definition & Clinical Settings

- Plasma Na > 150 mEq/L
- Less frequent than hyponatremia.
- Because thirst obligates patients to drink water that relieves the hypernatremia with only a 1 to 2% rise in plasma osmolality.
- Thus patients with water losses generally do not develop hypernatremia unless there is a defect in thirst mechanism or the patient is unable to get his needs of water
- The very young, the very old and the very sick are those who may be liable

Sings & Symptoms of Hypernatremia -1

- Polyuria and polydipsia may be due the underlying urinary concentrating defect rather than the hypernatremia itself
- Cellular dehydration due to ECF hyperosmolality affects mainly the CNS

Rate of Development: Acute is more serious than chronic;

Age of Patients: Old pts are more vulnerable than young

Acute + Adult = 75% mortalilty

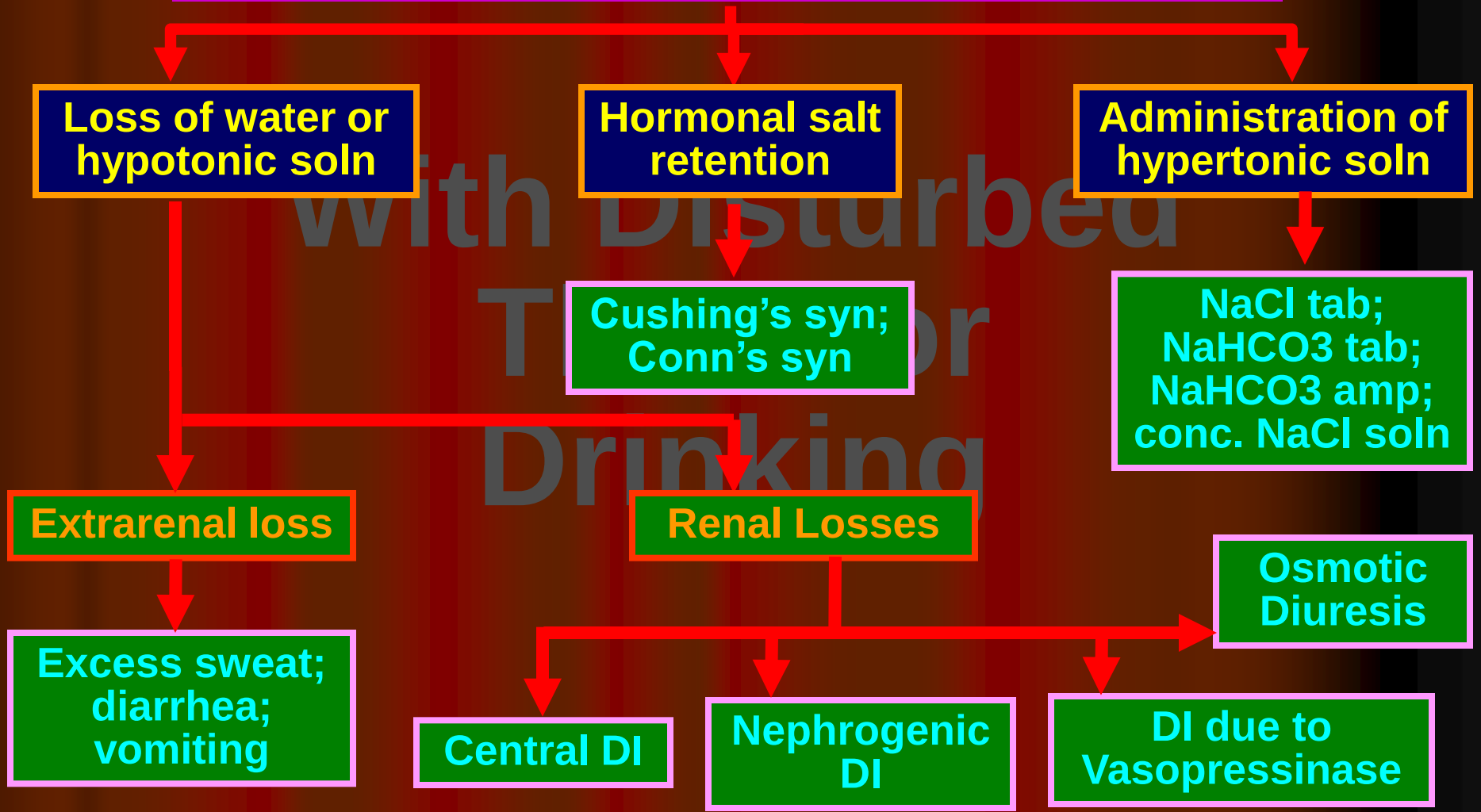
Acute + Children = 45% mortality

Sings & Symptoms of Hypernatremia -2

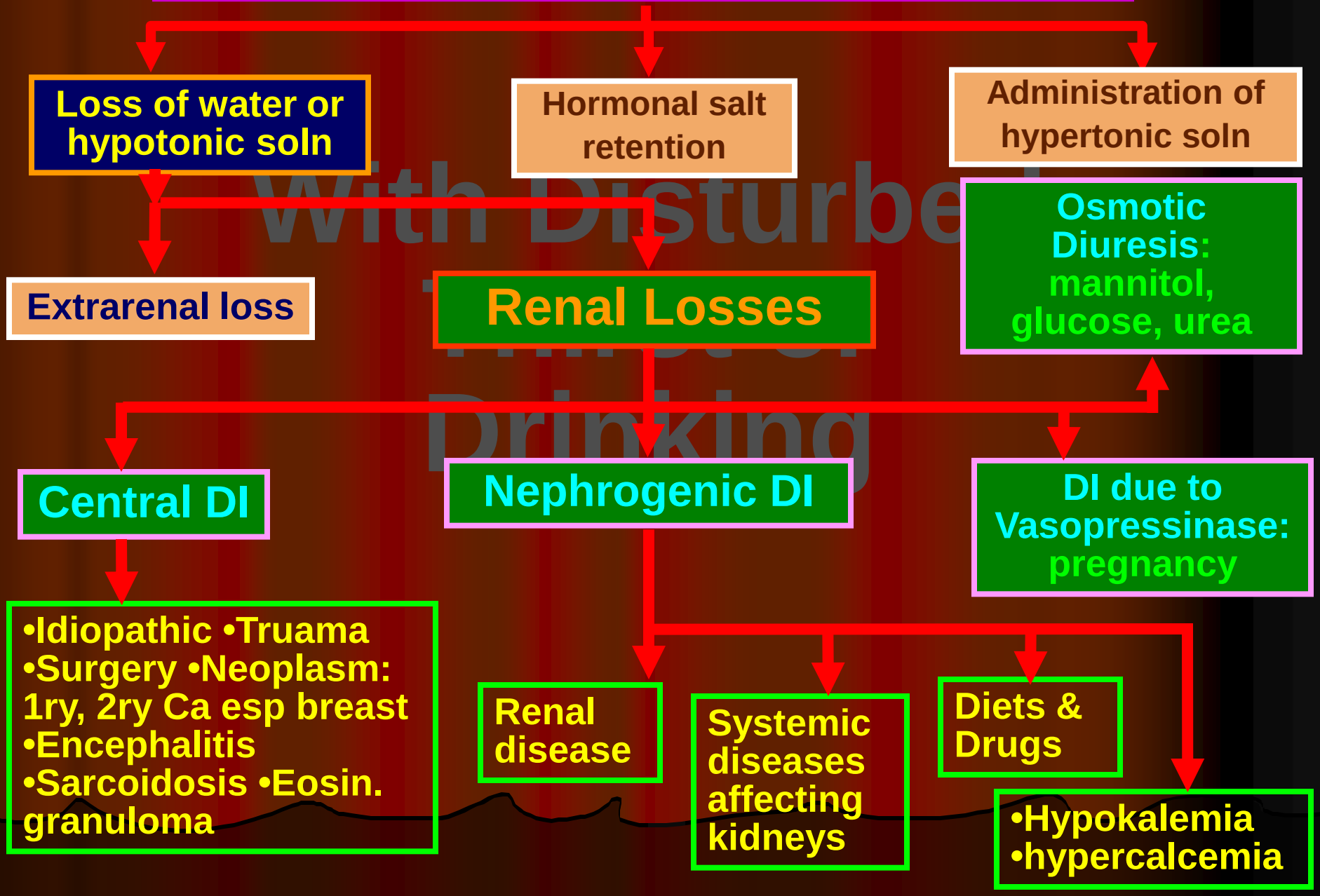
- Plasma osmolality > 325 mosm/Kg
- Shrinkage of brain cell
- Tearing of cereb vessels
- Capillary & venous congestion
- Subcortical & subarach. Bleeding
- Venous sinus thrombosis

- CNS dysfunction correlats with degree of hyperosmolality
- Restlessness, increased irritability, lethargy
- Muscle twitches, hyperflexia, tremulousness & ataxia
- Above 375 mosm/Kg: tonic muscular spasticity, focal & gand mal seizures

Causes of Hypernatremia -1



Causes of Hypernatremia -2



Nephrogenic DI

Primary Renal disease

- Medul cyst dis;
- CIN; • PCK;
- Part. Obstr.;
- CRF; • Polyuric ARF

Diet

- Very low Salt;
- Very low protein

Drugs

- Lithium; • Democlocyclin;
- Acetohexamide; • Glyburide;
- Colchicin; • Tolazamide;
- Propoxyphene

Systemic diseases affecting kidneys

- Multiple myeloma;
- Amyloidosis;
- Sarcoidosis;
- Sjogren's disease

Diagnostic Approach to Hypernatremia

Hypovolemia; TBW ↓↓; TBNa ↓

- **U.Na >20; U.Osmo: ↔ or↓**
  - **Ren. losses:**
    - Osm. Or loop diuret,
    - post-obstruction;
    - Intrin. renal disease
- **U.Na < 10; U.Osmo: ↑**
  - **Extra-renal losses:**
    - Insens. losses;
    - GIT losses

Euvolemia; TBW ↓; TBNa ↔

- **U.Na variable U.Osmo: variable**
  - **Ren. losses:**
    - DI: central, nephro., partial, gestational
    - Hypodipsia
- **U.Na variable U.Osmo: ↑**
  - **Extrarenal losses:**
    - Insens. loss: respirat., dermal losses

Hypervolemia; TBW ↑; TBNa ↑↑

- **U.Na >20; U.Osmo: ↔ or↑**
  - **Na gain:**
    - 1ry Hi-Ald;
    - Cushing's
    - Hi-tonic Dx
    - Hi-ton. NaCl
    - $NaHCO_3$ tab

Management of Hypernatremia

Hypovolemia

- Treat hypovolemia first by N saline

Euvolemia

Hypervolemia

- Treat hypervolemia first by diuretics ± dialysis if RF

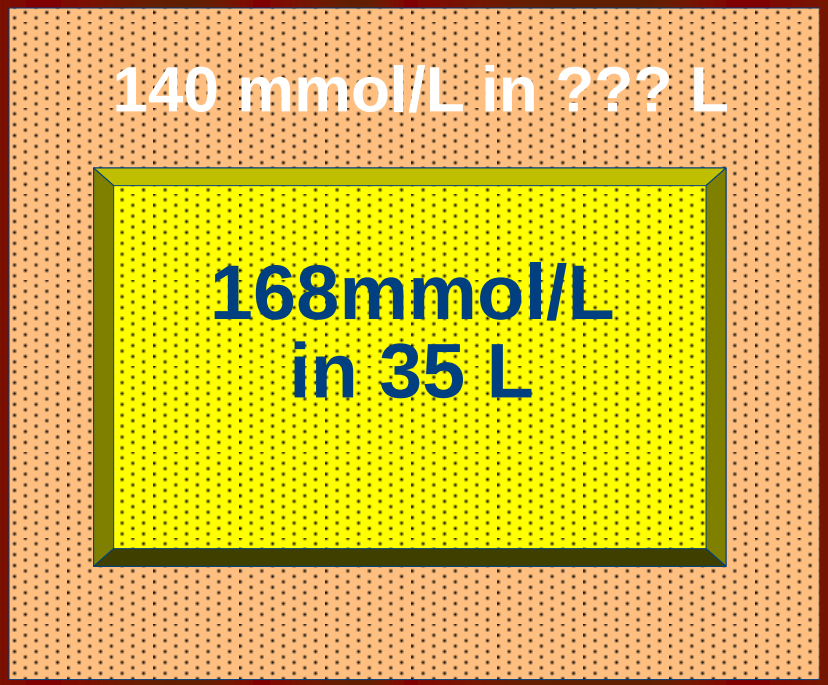
Treatment of the cause

- Correct hypernatremia by Water replacement: oral water or IV 5% Dextrose.
- Amount to be replaced to incr. Na 10 mmol/L = 0.04 X BWt

Rate of Correction: In acute cases: rapid correction, while in Chronic cases < 2.0 mosm/h or 1/2 correctn over 24 hs & 1/2 correction over next 24 hs

Correction of Water Deficir

W1 X Na1 = W2 X Na2



W1 = 35 L, Na1 = 168

W2 = ???, Na2= 140

= 35 x 168/140

= 42 L

Δ W = +7 L

Serum Potassium Disorders

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Physiological Considerations-1

- Average diet contains~100 mEq daily; 90% of which is excreted by the kidney. Normal serum K^+:3.5-5.5 mEq/L
- Hypokalemia and hyperkalemia are Common in the practice of medicine
- K^+ is present in the body in a larger IC (90%) and a smaller EC (10%) pools that are in series with each other
- In potassium-depleted states with normal acid-base status, a 1 mEq/liter fall in the S.K^+ level reflects the loss of about 300 mEq of K^+.
- Conversely, if large amounts of K^+ are administered acutely, the rise in S.K^+ level is less than would be expected if the administered K^+ were distributed solely in the ECF.

Physiological Considerations-2

Factors Affecting Transcellular Shift of K^+

Active transport processes:

- **Na^+-K^+-ATPase: actively transport K^+ into cell**
- **Insulin: promotes K^+ transport into cell**
- **β-adrenergic agents: promotes K^+ transport into cell**
- **Mineralocorticoids: promotes K^+ transport into cell**

Passive transport processes:

- **pH of ECF: alkalosis → intracellular K^+ shift, while acidosis → extracellular K^+ shift**
- **Increased ECF osmolality → extracellular K^+ shift**

Physiological Considerations-3

Effect of pH changes on transcellular shift of K^+

As a general rule, a reduction in plasma pH of 0.1 unit in metabolic acidosis raises the serum potassium level by ~0.5 mEq per liter, whereas a plasma pH increase of 0.1 unit produces a similar reduction in serum potassium.

0.1 unit Δ of pH $\rightarrow$ 0.5 mEq/L inverse Δ of $S.K^+$

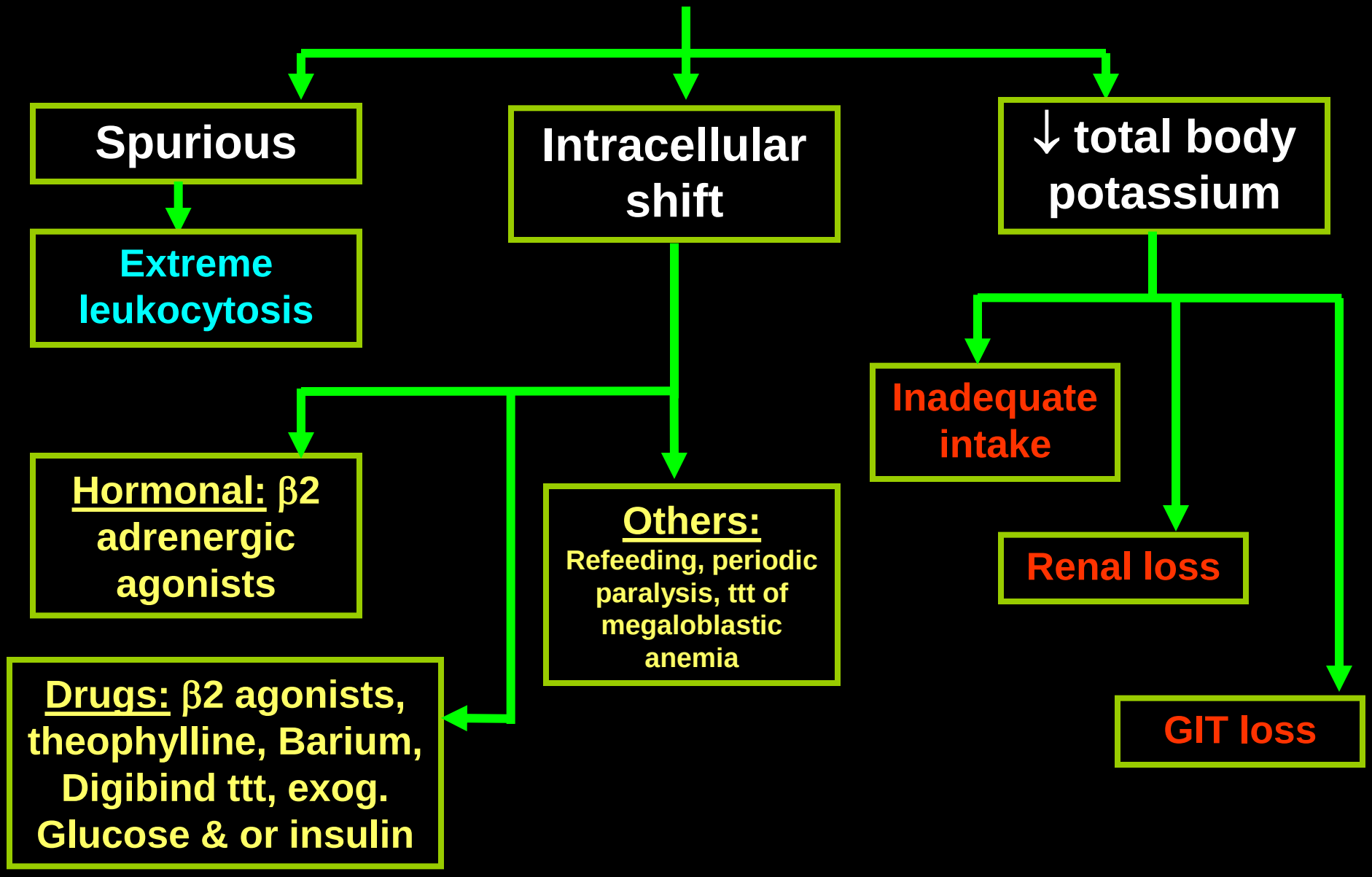
Physiological Considerations-4

Renal Handling of K^+

- ~90% of dietary K^+ is excreted by the kidney, while <10% is excreted by the GIT
- Almost all the K^+ excreted in urine gains access to the urinary space by secretory mechanisms located across distal convoluted and collecting duct segments.
- Factors causing increased urinary loss of K^+ are:
  - ↑ mineralocorticoids
  - ↑ delivery of Na^+ to collecting ducts
  - ↑ fluid flow to distal tubules
  - Metabolic and respiratory alkalosis
  - ↑ excretion of nonreabsorbable solutes

HYPOKALEMIA

Hypokalemia:Causes



Decreased Total Body K^+

Renal Loss

- Mineralocorticoid or glucocorticoid excess
- Bartter's syndome
- Thiazide, loop, & osmotic diuretics
- Renal tubular acidosis
- Chronic metabolic alkalosis
- Liddle's syndrome, acute leukemia, uretrosigmoidostomy

Extrarenal Loss

- Overt diarrhea
- Copious drainage from a fistula
- Villous adenoma
- Intractable vomiting
- ? Loss in perspiration
- Anorexia nervosa or tea & toast diet

Clinical Manifestation of Hypokalemia

Cardiac:

- Abnormal ECG
- Atrial & ventricular arrhythmias
- Predispose to digitalis toxicity

Endocrinal:

- Decr. Renin & Aldosterone
- Decr. Insulin secretion →diabetes
- Incr. Prostaglandin?

Hemodynamic:

- Variable BP
- Decr. pressor response to Ang.II

Kidney:

- Decr. GFR & RBF
- Polyuria & polydypsia: conc. defect + stimulate thirst
- Incr. Renal NH3 production: Hep. enceph
- Na+ retention
- Cl^- wasting
- Metabolic alkalosis

Neuromuscular:

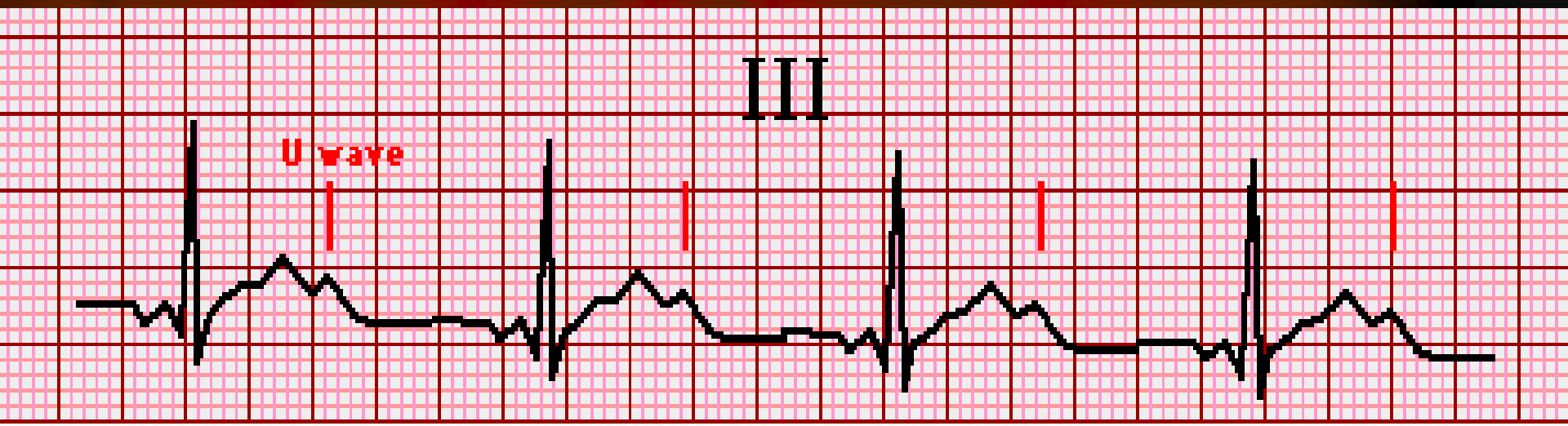
- GIT: constipation, ileus
- Skeletal ms: weakness, paralysis, rhabdomyolysis, respiratory paralysis

Hypokalemia + Metabolic Alkalosis:

- Alkalosis as a cause of hypokalemia
- Thiazide or loop diuretics
- Mineralocoriticoid or GC excess
- Excess vomiting
- Bartter's syndrome
- Mg depletion

Hypokalemia + Metabolic Acidosis:

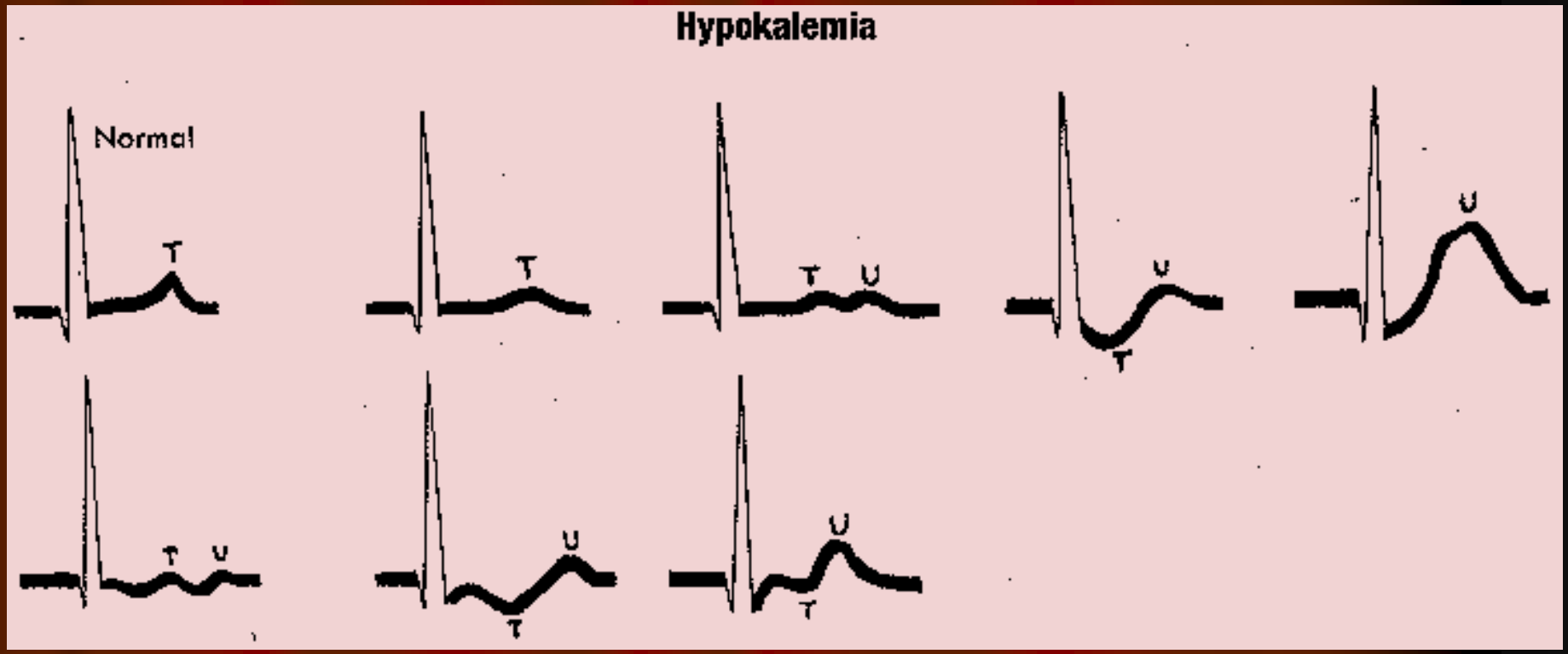
- Diarrhea
- Diuresis with CA inhibitors
- Renal tubular acidosis type 1 and 2
- Ureterosigmoidostomy

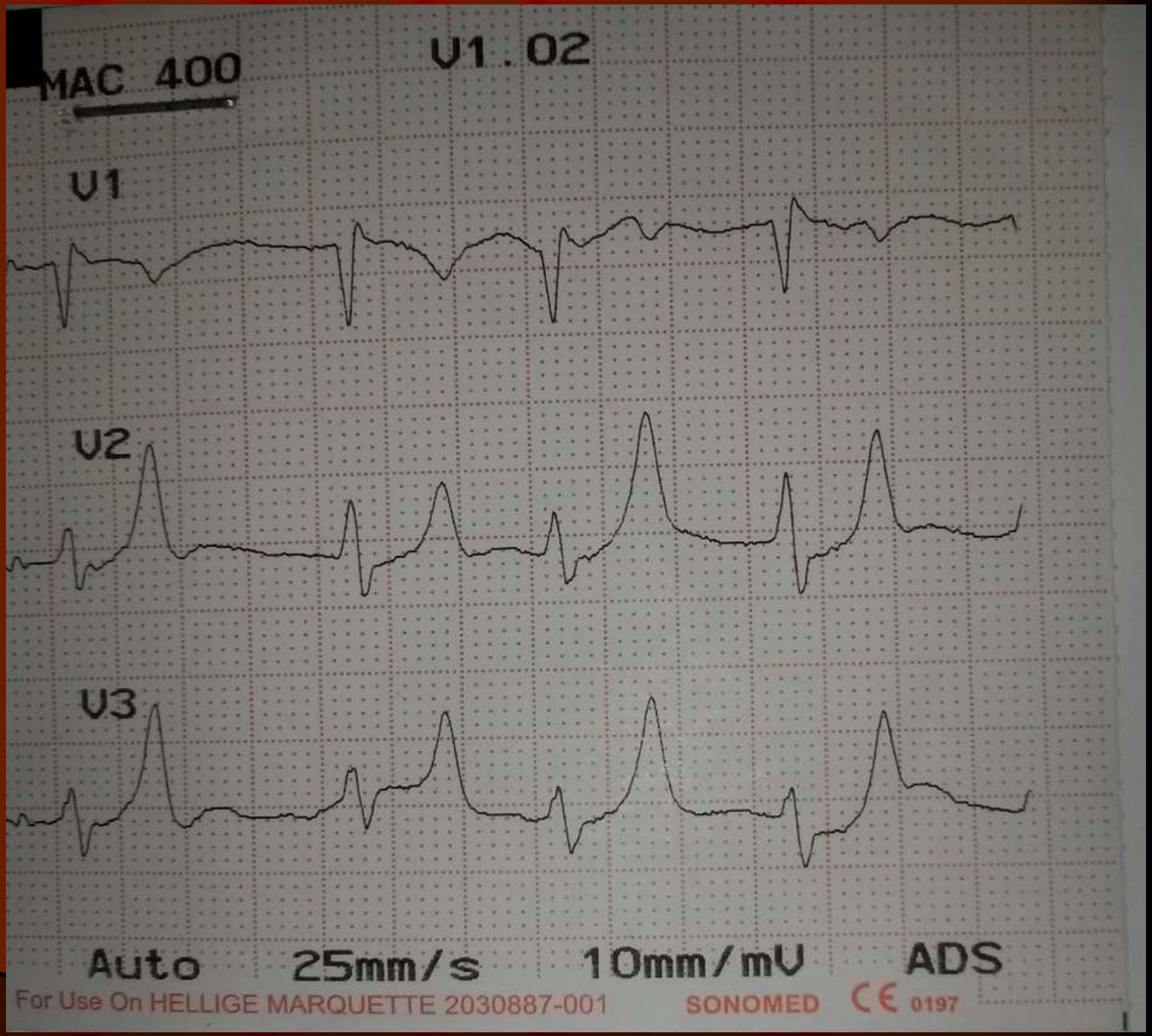


Hypokalemia An increase in the amplitude of U waves, which occur at the end of the T wave, are characteristic of hypokalemia.

Hypokalemia, continued

- ECG changes in hypokalemia




MAC 400
V1.02
V1
V2
V3
Auto 25mm/s 10mm/mV ADS
For Use On HELLIGE MARQUETTE 2030887-001
SONOMED
CE 0197

Management of Hypokalemia - 1

Estimation of K^+ Deficit

S. K+	Level	K^+ deficit	ECG changes
3.5 - 3.0	mild	100-200	No
3.0 - 2.5	moderate	200-400	variable
2..5 - 2.0	severe	400-800	dangerous

Management of Hypokalemia - 2

Route of K^+ Administration:

- **Oral route is preferred**
- **I.V. route is used in severe conditions**

Rate of K^+ Administration:

- **100 - 250 mEq/day :**
  - **in mild cases: 100**
  - **in moderate cases: 200**
  - **in severe cases: 400 mEq/day**
- **For parenteral route 10-30 mEq/hour: regular, slow and steady to allow equilibrium across cell membrane**

Management of Hypokalemia - 3

Type of K^+ salt:

- **KCl for alkalosis; K gluconate, acetate or citrate for acidosis; K phosphate for DKA**

Drugs for hypokalemia:

- **Potassium sparing diuretics: spironolactone, ameloride and triametrene**
- **ACE inhibitors**
- **Angiotensin receptor blockers**
- **Beta adrenergic blockers**
- **Cyclsporine & Trimethoprim**

HYPERKALEMIA

Hyperkalemia:Causes

Pseudo-hyperkalemia

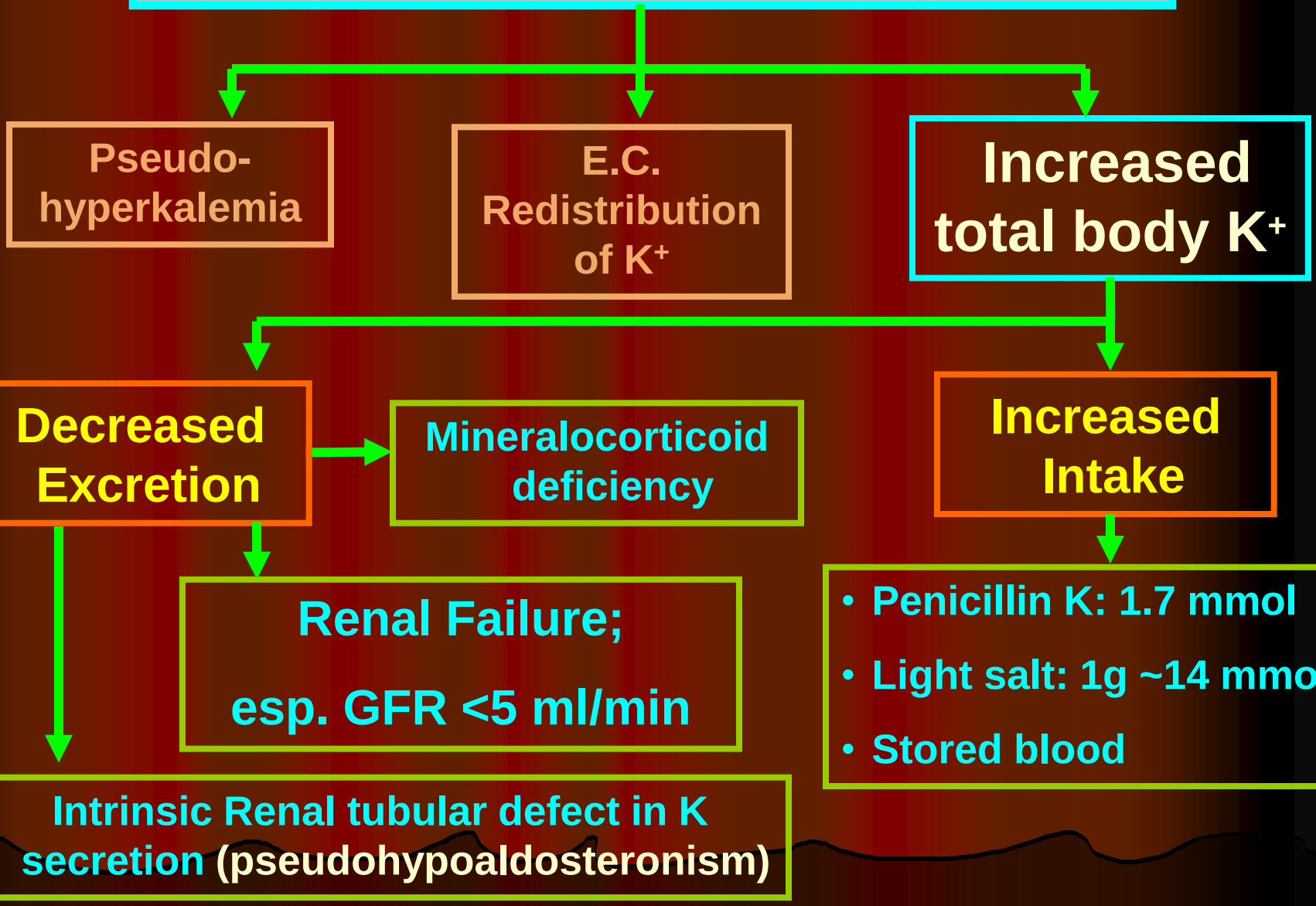
- Hemolysis
- Leukocytosis
- Thrombocytosis
- Exercise + ischemia of z limb

E.C. Redistribution of K^+

- Acidosis esp. hyperchloremic
- Insulin deficiency
- Hypertonicity e.g. glucose or mannitol
- Drugs: - beta blockers - cationic a.a. - Succinyl choline - Digoxine
- Hyperkalemic periodic paralysis

Increased total body K^+

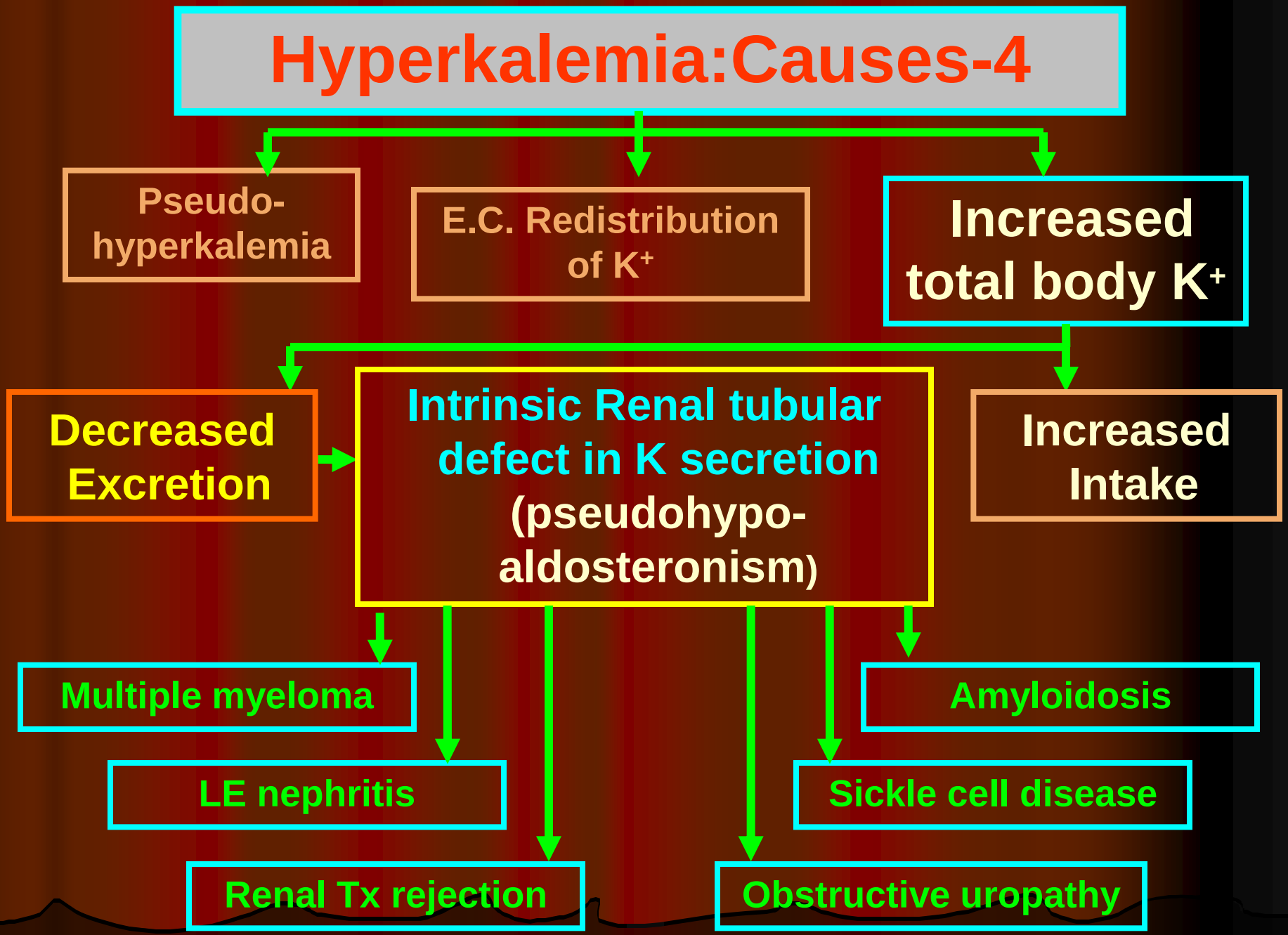
Hyperkalemia:Causes-2



Hyperkalemia:Causes-3

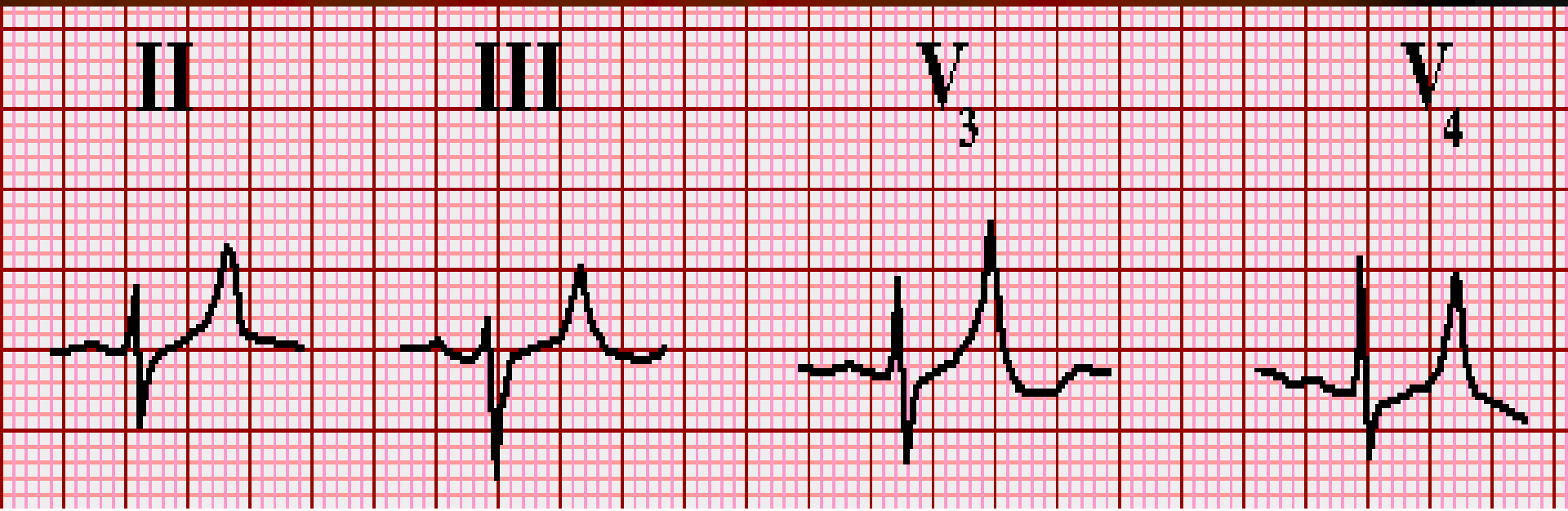
- Pseudo-hyperkalemia
- E.C. Redistribution of K^+
- Increased total body K^+
  - Decreased Excretion
    - Mineralocorticoid deficiency
      - Addison's disease
      - Hypoaldosteronism:
        - Hyporeninemic: DN, CIN
        - Hyperreninemic: resistance to aldosterone
    - Drugs:
      - ACEIs
      - NSAIDs
      - Heparin
      - Cyclosporin
  - Increased Intake

Hyperkalemia:Causes-4



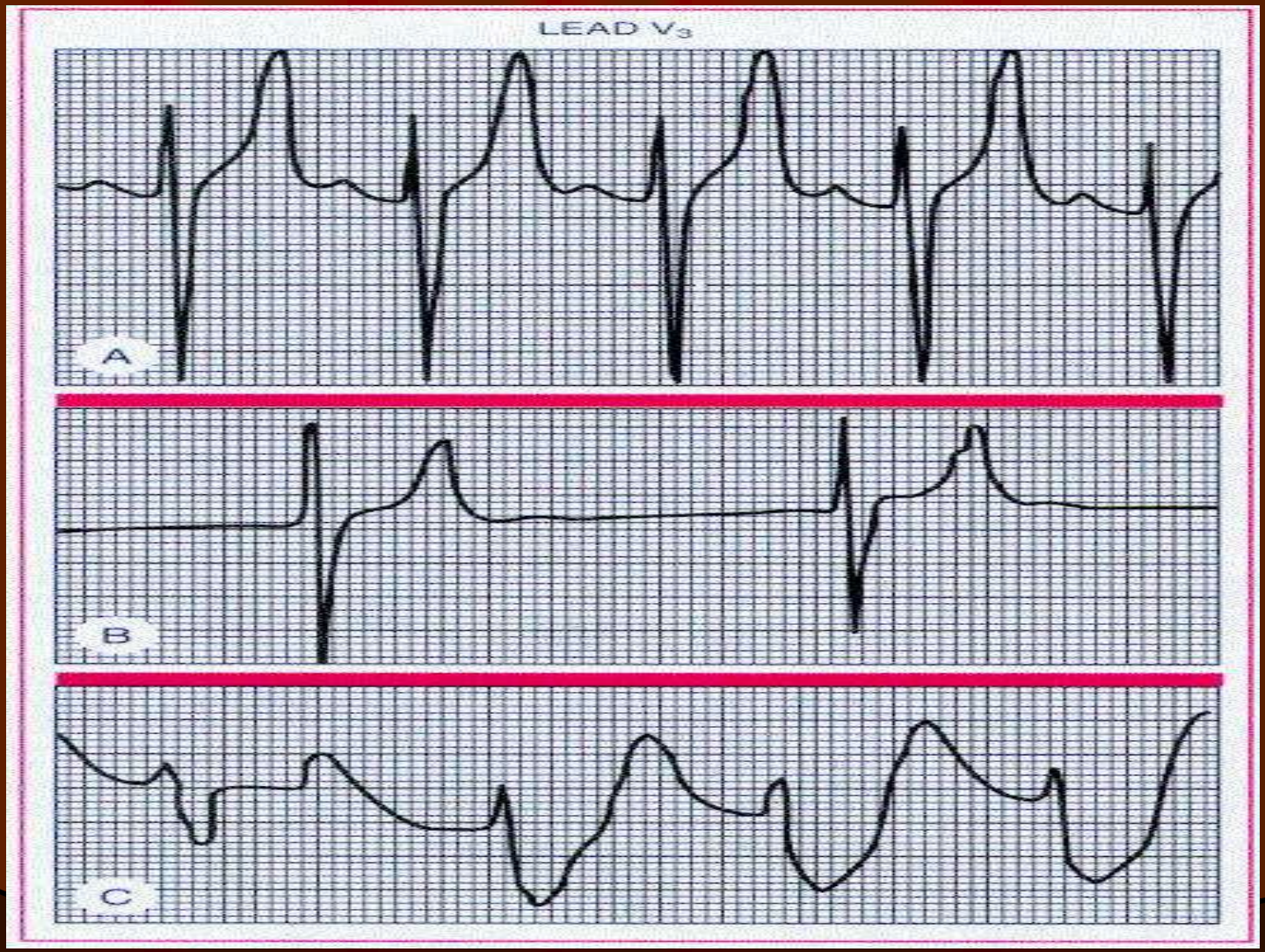
Signs & Symptoms of Hyperkalemia

- Cardiac conduction effects with potential cardiac arrest
- ECG changes correlate to some extent with the degree of hyperkalemia
- Neuromuscular symptoms include tingling, parathesia, weakness and even flaccid paralysis
- Cardiac toxicity usually precedes other manifestations
- Hyperkalemia stimulates aldosterone, insulin, and glucagon secretion and suppresses plasma renin



Hyperkalemia A tall peaked and symmetrical T wave is the first change seen on the ECG in a patient with hyperkalemia.

Hyperkalemia – ECG Changes



Management of Hyperkalemia - 1

Accord. to presence ECG changes or paralysis

Urgent ttt

- **Immediate onset 1-3 min:** 10-30 ml 10% Ca-Gluconate IV
- **Quick onset 5-10 min:** 25g IV glucose + 5-10 U sol insulin
- **Quick onset 15-30 min:** 50-150 mEq $NaHCO_3$ IV
- **Quick onset 15-30 min:** Albuterol 20 mg in 4 ml nebulizer

Conservative ttt

- Decr. Diet K
- withdraw offending drugs
- Drugs that incr. K excretion: lasix, NaCl, K-exchange resins
- Treatment of cause
- Dialysis or Tx if RF